ventricular septum (n=1), right ventricular free wall (n=1) and coronary cusp (n=1). Acutely 16/26 (62%) ablations were successful, 3/26 (12%) partially successful (arrhythmia became non sustained or there were other arrhythmias that were not targeted and 7/26 (27%) unsuccessful. Three of the unsuccessful ablation sessions were repeated within 3-20 days. There were 3 complications (1 respiratory distress, 1 cardiac perforation and 1 heart block). There was no recurrence at follow-up in 13/21 (62%) patients (mean follow up time 304.0±268.3 days). Time to recurrence (documented ventricular arrhythmia) in 8/21 (38%) patients was 137±158 days. There were 2 deaths at 3 and 34 days (1 due to ventricular arrhythmia and 1 due to cerebrovascular accident).

CONCLUSION: Although infrequent catheter based ablation for ventricular tachycardia in patients with NIDCM is relatively safe and has good short and long term success rates.

1063-138 Smoking Is Independently Associated With Cardiomegaly In Coronary and Non-Coronary Deaths in Men

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Smoking and left ventricular hypertrophy are each independent risk factors for sudden cardiac death. Although coronary artery disease and hypertension are known to cause cardiomegaly, an independent relationship between cigarette smoking and increased cardiac mass has not been documented. We studied 462 hearts at autopsy of men dying unexpectedly due to severe coronary atherosclerosis (n=307, 52 ± 11 years old) and non cardiac causes (n=155, 42 ± 13 years old). Cases were selected prospectively, excluding genetic causes of cardiomegaly, idiopathic dilated cardiomyopathy, pure right-sided hypertrophy, and valvular heart disease. Body weight, height, estimated body mass index (BMI, kg/m2), smoking (by history and corroborated by post-mortem serum thiocyanate determination), diabetes (by history and blood glycohemoglobin determination), hypertension (by history and real microvasculature assessment) and total cholesterol/high-density lipoprotein cholesterol ratio were determined at autopsy, as well as an estimate of coronary plaque burden (sum of maximal % luminal narrowing of 4 major epicardial arteries). By univariate analysis, smokers dying with severe coronary disease had increased BMI-adjusted heart weight (493±119 vs. 468±110 grams for non-smokers, p=.01), as well as control smokers (452 ± 116 vs. 410 ± 76 grams for non-smokers, p=.01). By multivariate analysis in atherosclerotic deaths, smoking (p=.0003) was independently associated with increased heart weight, independent of body mass index (p=.0001), age (p=.0001), hypertension (p=.01), and plaque burden (p=.03); race, cholesterol, diabetes, and history of alcohol or drug abuse were not significantly associated with heart weight. Smoking was also independently associated with heart weight in controls (p=.009), independent of hypertension (p=.001), age (p=.001) and body mass index (p=.0001); other parameters were not significantly associated with heart weight. These results suggest that smoking is a risk factor for cardiomegaly, in addition to hypertension, obesity and coronary artery disease. The mechanism by which smoking increases the risk of sudden death may in part be mediated by ventricular hypertrophy.

1063-139 Influence of Gender and Alcohol on Mortality in Nonischemic Dilated Cardiomyopathy

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Background: Alcohol is a major cause of cardiomyopathy in Western Societies. In contrast to ischemic heart disease, little attention has been directed towards the identification of the effect of alcohol intake on mortality in patients with non-ischemic heart failure.

Objectives: This study sought to examine the association between reported alcohol intake and subsequent cardiac mortality and whether the effect of alcohol is modified by gender in patients with non-ischemic dilated cardiomyopathy (DCM).

Method and Results: Between 1994-1998, we retrospectively studied a cohort of 396 consecutive patients with DCM, 74% men, mean age 53 ± 15 years. The history of alcohol intake was abstracted from the medical records. During a subsequent follow up period of 48 ± 36 months, 83 (76% men) patients died and 15 (80% men) underwent cardiac transplantation. Men were younger and more likely to have a history of excessive alcohol intake compared to women. Gender significantly modified the risk of alcohol consumption on mortality. Whereas alcohol intake increased the mortality risk in women (hazard ratio for death (HR), 4.2, 95% confidence interval (CI), 1.4-9.3; p = 0.009), it was protective towards death in men (HR, 0.42, 95% CI, 0.37-1.0; p = 0.05). The effect of alcohol intake on outcome was reassessed again after classifying the patients into 4 groups: group 1 life-long non-drinkers, group 2 former drinkers, group 3 and 4 current drinkers. Moderate drinking was defined as <14units/week for women and <21 for men. Heavy drinking as >14 and >21 units a week, respectively for women and men. The 5-year survival rates for the groups among women and men were different; 90%, 90%, 63%, and 56%; p = 0.03 vs 67%, 90%, 80%, and 60%; p = 0.02.

Conclusion: In our study, we have demonstrated that the risk of reported alcohol intake on mortality is related to gender in patients with non-ischemic heart failure. Although women live longer, they are more sensitive to the toxic effects of alcohol than men. Our findings deserve further prospective study as a possible way to improve outcome in such patients.

1063-140 Arrhythmias in Biopsy Proven Acute Myocarditis Versus Acute Pericarditis With No Evidence of Myocarditis in Endomyocardial Biopsy

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Background: It is controversial whether the arrhythmias in acute pericarditis are of myocardial or pericardial origin.

Methods: The aim of the study was to investigate the ECG changes, conduction, and cardiac rhythm disorders in endomyocardial biopsy (EMB) proven acute myocarditis/perimyocarditis (Group 1: 10 pts, 3/10 with perimyocarditis, 70% males, mean age 46.1±15.8 years, mean heart rate (HR) 76.7±33.1 b/min) in comparison to acute pericarditis with no EMB evidence of myocarditis (Group 2: 40 pts, 65% males, mean age 45.6±15.7 years, mean HR 98.7±22.2 b/min). At the initial assessment all pts underwent echocardiography, cardiac catheterization, and biventricular EMB. Tissue samples were independently reviewed by two cardiac pathologists. Immunohistochemistry and immunocytochemistry were performed, and only pts fulfilling Dallas and WHF criteria were selected for Group 1.

Results: Comparative analysis of ECGs and 24h Holter recordings at initial presentation is shown in the table. Paroxysmal supraventricular tachyarrhythmias (PSVT), and ventricular fibrillation (VF) were significantly more frequent in myocarditis (one pt died and 2 underwent ICD implantation) in contrast to atrial fibrillation (AF)(all p<0.05) .

Conclusion: In pts with EMB proven acute myocarditis/perimyocarditis life threatening rhythm disorders occurred significantly more often than in pts with pericarditis with no EMB indications of myocarditis, which on the contrary had a higher incidence of AF.

(%)	AF	Low Voltage	ST/T Changes	AV Block Gr. II	AV Block Gr. III	PSVT	VT	VF
Myocarditis (N=10)	0*	30	30	0	10	40*	10	20*
Pericarditis (N=40)	20	40	47.5	5	2.5	5	2.5	0

1063-161 The Coxsackie-Adenovirus Receptor Is Associated With Differential Susceptibility to Group B Coxsackieviral Infection and Myocarditis

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BACKGROUND: Group B coxsackieviruses (CVBs) are a common etiologic agent of viral myocarditis and dilated cardiomyopathy. The expression of specific viral receptors, such as the coxsackie-adenovirus receptor (CAR), may be the primary determinant of host susceptibility and organ tropism.

METHODS: To explore CAR as a determinant of myocarditis susceptibility, the pathogenesis of CVB3 infection was investigated in A/J and C57BL/6 mice. The organ distribution of murine CAR (mCAR) RNA and protein was compared between strains. In addition, mCAR was cloned and sequenced systematically to screen for mutations derived from murine tissues, cardiomyocytes and cell lines.

RESULTS: Following CVB3 infection, survival was similar between strains, but on histopathological examination, myocarditis was more severe in A/J mice. Using an RNA probe for CAR it was found that mCAR RNA expression was highest in liver, heart and kidney. Lower expression was detected in the pancreas, spleen and brain. CAR protein expression was highest in the hearts of the myocarditis-susceptible A/J mice. This is in association with increased CVB3 replication in A/J hearts. Sequencing of cDNA clones from A/J myocytes and Vero cells led to the identification of a truncated CAR isoform, highly homologous between murine and monkey cells. The transmembrane and cytoplasmic regions of full length mCAR1 are replaced by a 24 amino acid segment at the C-terminal end of the protein. In addition, a recurring amino acid substitution in the first immunoglobulin loop (D1) of the extracellular portion of mCAR2 was identified. The ability of truncated CARs to mediate virus attachment was demonstrated by CVB3 binding assays. But, interestingly, productive infection was not observed.

CONCLUSION: In conclusion, increased mCAR expression, and differential expression of receptor isoforms may play a role in susceptibility to CVB3 infection and myocarditis.

1063-162 The Natural History of Restrictive Cardiomyopathy Presenting During Childhood

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Background: Restrictive cardiomyopathy (RCM) in children is associated with a poor prognosis. A better understanding of the natural history, especially the time to deterioration, would facilitate management.

Methods: The National Australian Childhood Cardiomyopathy Study is a population-based study of all children in Australia with primary cardiomyopathy (CM) who presented at 0-10 years of age between 1987-97. Diagnostic criteria was elevated filling pressures at cardiac catheterization and/or evidence of reduced diastolic ventricular volume with atrial enlargement on echocardiography, in the presence of normal ventricular wall thickness and preserved systolic function. Serial echocardiographic measurements were expressed as Z scores. Study end-points were death or transplantation.

Results: There were 8 patients with RCM, representing only 2.5% of the study population. Half presented with congestive heart failure and the other half with progressive exercise intolerance. The median age at presentation was 3.05 years (range: 1.3-7.6) with at least 50% of all other CM types presenting less than 6 months of age (p=0.003). One child had a positive family history for CM and none had biochemical evidence of metabolic disease. All patients had biventricular restrictive physiology. From presentation there was a decline in mean left ventricular (LV) end diastolic dimension (Z score +0.22 to -0.13) and mean LV fractional shortening (Z score +0.48 to -2.1), with an increase in mean LV posterior wall thickness (Z score -0.82 to +0.2). At latest follow-up 4 (50%) have died, 3 (38%) have undergone cardiac transplantation and 1 (12%) remains in chronic right heart failure. Freedom from death or transplantation was 75% at one year after presentation and 38% at 5 years. The median time to death or transplantation was 2.39 years (range 9 days-8.49 years).

Conclusions: RCM presenting in children is rare, manifests with biventricular involvement and presents at an older age compared to other CM types. Although the eventual outcome is poor, survival in this population-based study suggests that urgent cardiac transplantation is seldom required at the time of presentation.